age in which repeated rheumatic infections are most likely to occur. Following the above criteria it is hoped that the operative mortality will diminish in time and that properly selected heart patients will, in the future, derive benefit from surgery.

THE SURGICAL RELIEF OF CARDIAC PAIN

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WILDER PENFIELD

The operation of cervical sympathectomy has in the past been used to treat glaucoma, exopthalmic goitre, migraine and other maladies. We have consigned such treatment of these conditions to that large and ever growing heap of discarded remedies. Those of us who are using this operation in the treatment of angina are in danger of seeing this treatment also go into scornful discard, unless we are guided in our application of the remedy by a reasonable rationale. I am not qualified to judge of the effect of these operations upon heart action. Those cases upon which I have operated at the Presbyterian Hospital have been studied from this point of view by Dr. Robert Levy. The two cases to be reported tonight throw new light upon the nervous mechanism involved and make possible what would seem a more satisfactory explanation of the nervous processes involved in angina pectoris.

The sympathetic nervous system has three direct connections with the cardiac plexus, the superior, middle and inferior cardiac nerves. All carry motor impulses to this plexus. The superior cardiac nerve probably carries most or all of the constrictor fibers to the coronary vessels and aorta, as urged by Ransom (7). The middle and inferior cardiac nerves are called by Langley (9) the minor and major accelerators of the heart. Although all three nerves carry motor fibers, only the lower two contain sensory fibers from cardiac plexus to sympathetic ganglia. These afferent impulses reach the central nervous system through the white rami to the upper six thoracic spinal segments. Afferent fibers also pass up the vagus.

Thus the rationale (or lack of it) for the various operations performed appears evident. Jonnesco (8) removed the lower part or all of the chain in order to eliminate all afferent paths through the sympathetic. Hoffer (6) cut a branch of the vagus which he believed to be the depressor, to eliminate vagus afferents, and Coffey and Brown (1) removed the superior sympathetic ganglion because it was easily accessible. Ransom has suggested that this last operation may prevent reflex spasm in the coronaries and aorta.

As I shall take up the following cases only from a neurological point of view, it is sufficient to say that the first patient, G. C., had a syphilitic aortitis with aortic insufficiency. The angina pectoris was severe and completely incapacitating. It began in the manubrium and spread to the inner aspect of the left arm. If not checked by nitroglycerine it then spread to the inner aspect of the right arm.

According to the theory of referred pain as first suggested by Head (5) and Mackenzie (11) a stimulus passes from the heart via the sympathetic to a posterior root ganglion. Here in some way the sympathetic impulse is supposedly turned into stimulation of certain spinal ganglion cells which cause the patient to feel pain. This pain he refers to the sensory distribution of the nerve root in question.

According to this theory, it should have been sufficient to remove the middle and inferior cervical and stellate ganglia on both sides. This of course interrupts all sympathetic connection between cardiac plexus and central nervous system. Consequently, this operation was performed on both sides leaving in place both superior cervical ganglia. It is evident that each superior sympathetic ganglion is now isolated. Its only connection with the spinal cord which had been by means of the sympathetic chain is now removed. It is provided only with efferent outlets to the vessels of the brain, the outgoing branches of the fifth nerve, the peripheral distribution of the highest three or four cervical spinal nerves and the cardiac plexus. It cannot be the path for sensory conduction—first, because there are apparently no sensory fibers in the superior cardiac and secondly because the ganglion now has no known connection with the central nervous system.

Nevertheless, on the ninth day after operation the patient sent for the interne on the ward because of pain in his head and the following is Dr. Samuel Lambert's careful note made at the time: "The patient was sitting straight up in bed. His forehead was moist with perspiration equally on both sides. The rest of his body was warm but not moist. He was perfectly clear mentally. He said he had a dull pain which started all around his neck, just below his jaw, like a collar. It went up into his gums and then up the sides of his head to the top. At the same time he had a feeling of tightness in exactly the same locations. He had no pain or feeling of constriction any other place. His blood pressure was over 300 systolic and 0 diastolic. He felt a little nauseated momentarily.

"He was given 0.0006 gms. nitroglycerine. After five minutes he said the pain had entirely left. In the patient's words, 'It just kind of floated away.' His blood pressure was still over 300 systolic and 0 diastolic.

"Fifteen minutes later he said he had a very slight pain returning in his lower jaw. Blood pressure still over 300 and 0. No perspiration on forehead. Pulse throughout 82–86.

"One half hour later he was given a second dose of nitroglycerine. Pain completely gone. Blood pressure 170 systolic and 0 diastolic."

It is now a little over a year since the operation and although he was restored to activity again, he has continued to have attacks of angina pectoris in the face, head and sometimes neck. These attacks are easily relieved by nitroglycerine.

It is impossible to explain these seizures on the basis of referred pain. The angina which the patient experienced was in the distribution of the fibers of the superior sympathetic ganglion which accompany the branches of the fifth nerve and upper three cervical nerves, but this ganglion is cut off from the central nervous system. Therefore, I have assumed that the ganglion is stimulated by a reflex in the autonomic system. Elsewhere in the sympathetic system axone reflexes (10) have been clearly recognized and Gaskell has stated that by analogy he should expect the vagus to enter into such reflexes (4). Axones in the vagus may carry the impulse by connectors through the

rich anastomosis to the ganglion. The motor cells of the ganglion discharge and cause spasm of the smooth muscle in the vessels and sweat glands which they innervate. This peripheral spasm is then appreciated as the pain of angina through the pathway of the cerebro-spinal nerves. Instead of the pain being referred it is actually felt in the periphery. As was mentioned above nitroglycerine which relaxes smooth muscle, stopped the pain, sweating soon ceased and the blood pressure came down.

The second patient to be described, S. A., aged 38, had a general arterio-sclerosis and hypertension, his blood pressure averaging about 210–105. Walking across the ward was sufficient exertion to precipitate a severe attack of angina pectoris, felt as pressure in the precordium and left arm.

I removed the left cervical sympathetic chain, including all the cervical ganglia and the stellate ganglion, on the advice of Dr. Levy. When the patient became active after the operation, typical attacks of angina pectoris reappeared. But now it was on the right side and the sweating which accompanied it was on the right side of the face and right arm. Some pressure was felt in the upper sternum, right side of the neck and inner aspect of the right arm. These attacks continued and he was again operated upon. This time, because of the good results reported by Coffey and Brown, the simple operation of removing the superior sympathetic ganglion was performed. On the third day after removal of the right superior ganglion, angina recurred. Now the pain appeared to be in the right arm, the right neck being left out, and sweating now, during attacks, was altogether absent in the face but appeared in the right arm and chest.

Thus, a second time we see that the angina was felt in the area where the sympathetic motor supply was intact. After the second operation the patient's systolic pressure fell from an average of 210 to 170. It rose, however, as was the case with the first patient during attacks (reaching 205). Recovery from the immediate effects of the operation was satisfactory but on the eighth day after operation the patient suddenly had what Dr. Levy felt to be a coronary occlusion, and he died on the sixteenth day.

On the theory of an axone reflex, the conditions are exactly the same as those in which these reflexes occur elsewhere in the sympathetic nervous system. By stimulation of sympathetic axones in the cardiac plexus, the motor cells in the sympathetic ganglia would be excited causing localized sweating, arterial spasm and some increase in blood pressure.

On the above basis certain phenomena of angina are easily explained. Mackenzie (12), when investigating the pilomotor (or goose-skin) reflex, found that rubbing lightly the skin under the left breast caused the goose-skin to pass up the left side of the chest and down the inner aspect of the left arm. This sympathetic reflex induced a chilly sensation in the same distribution. When he tried the reflex on a case of angina, the patient remarked in surprise that the chilliness corresponded with the usual distribution of his pain. This pilomotor reflex is due to a sympathetic motor discharge and the resultant sensation of chilliness is reported to the consciousness by way of the sensory spinal nerves. The mechanism is the same as has been suggested above for angina and the resulting sensation has the same peripheral distribution.

The hyperaesthesia which may follow a seizure would indicate that the pain was due to a peripheral condition just as any severe pain from a peripheral cause may leave a hyperaesthesia of the skin. Patients at times complain that pressure on some area of skin which may be quite small and well localized induces a bout of angina. Such pressure might perhaps set up smooth muscle contraction in an area already receiving subliminal stimulation.

That an angio-spasm originating in the stellate ganglion may be the cause of the pain in angina was suggested by Brünning (2) for the following reasons: (1) In handling the ganglion preparatory to removing it he observed that his patient's left arm became suddenly blue as in Raynaud's disease; (2) The fact that blood pressure rises during attacks.

The new hypothesis proposed for the explanation of the nervous mechanism in angina pectoris depends upon an autonomic axone reflex. It might well be called, therefore, reflex pain. It causes peripheral spasm of smooth muscle and thus may re-

semble the pain in various angiospastic conditions. It differs from the referred pain of head principally in that the point of contact of sympathetic and cerebrospinal system is shifted from the posterior root to the periphery where there are other analogous contacts between the two. No attempt has been made here to expand the hypothesis to other types of visceral pain nor to analyze different kinds of cardiac pain.

Finally certain practical conclusions may be drawn:

1. The removal of a sympathetic ganglion removes the possibility of angina pectoris in the *motor* distribution of that ganglion only.

2. Pain is still possible in the motor distribution of the remaining ganglia, provided the stimulus arising in the heart or aorta is adequate.

3. Success in the operation depends not upon interrupting a direct afferent path from cardiac plexus to central nervous system as has been assumed, but upon the interruption of autonomic reflexes.

4. Complete cervico-upper-thoracic sympathectomy should only be employed in cases where life is really insupportable even under the best medical care. In certain less severe cases it is possible that removal of the superior sympathetic ganglion may be of benefit because of the resulting effect upon the irritative focus in the heart or aorta.

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THE APPLICATION OF SURGERY IN THE TREATMENT OF MITRAL STENOSIS

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I. INTRODUCTION

For the privilege of addressing the Academy of Medicine of New York City I am very grateful. I should like to express my gratitude for this privilege to Dr. Elliott C. Cutler, Professor of Surgery at the Western Reserve Medical School. With the collaboration of Dr. S. A. Levine the application of surgical methods in the treatment of mitral stenosis was carried out at the Peter Bent Brigham Hospital and the Laboratory of Surgical Research of the Harvard Medical School. We are continuing studies in the subject at the Western Reserve Medical School. To our teachers, Dr. Harvey Cushing and Dr. Henry Christian, we take pleasure in expressing acknowledgment and indebtedness.

II. RATIONALE OF THE PROCEDURE

The fundamental idea upon which this work is based is that if the mitral obstruction be decreased the mechanics of the cir-